

Thoracoabdominal Compression Model of Traumatic Asphyxia to Identify Mechanisms of Respiratory Failure in Fatal Crowd Accidents

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Background: Traumatic asphyxia is a major cause of death in fatal crowd disasters, but the relationships between compression site, load magnitude, load time, and medical outcomes are unclear. This study estimated thoracoabdominal compression conditions (load magnitude, load time) that could result in respiratory failure in adults.

Methods: Eight load patterns-A (chest load: 0 kg, abdominal load: 10 kg), B (0, 20), C (10, 0), D (10, 10), E (10, 20), F (20, 0), G (20, 10), and H (20, 20)-were applied to 14 healthy women. Blood pressure, heart rate, respiratory rate, SpO₂, tidal volume, vital capacity, respiratory phase, and modified Borg dyspnea score were measured over time. Breathing Intolerance Index (BITI) was also calculated.

Results: Vital capacity decreased in patterns C, D, E, F, G, and H. BITI reached the critical range of ≥ 0.15 (at which respiratory failure occurs after about 45 min) after 14 min in pattern G and 2 min in pattern H. A vital capacity ≤ 1.85 L and a modified Borg scale score ≥ 8.3 corresponded to a BITI of ≥ 0.15 and were regarded as equivalent to reaching the critical range. Furthermore, change in chest load was positively correlated with BITI when abdominal load remained constant.

Conclusions: In women, respiratory failure can occur within 1 h from respiratory muscle fatigue, even when total thoracoabdominal load is only about 60% of body weight. A vital capacity ≤ 1.85 L and modified Borg scale score ≥ 8.3 can be regarded as indices for predicting respiratory failure.

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Key words: traumatic asphyxia, crowd accident, stampede, respiratory failure, Breathing Intolerance Index (BITI)

Introduction

Traumatic asphyxia is a major form of trauma caused by respiratory and circulatory failure due to severe external compression of the chest, such as when a person is caught in a machine, crushed in a stampede, or buried in sand. It results in death due to hypoxic encephalopathy^{1–5}. Traumatic asphyxia is considered the most common cause of death in human stampede accidents at sports and religious events worldwide and is character-

ized by petechial hemorrhage on the face, neck, and palpebral conjunctiva, due to obstruction of venous return, and absence of organic findings such as multiple rib fractures and thoracoabdominal organ injury^{5–13}.

During a human stampede at a fireworks display in Japan in 2001, eleven people were killed (9 children younger than 10 years and 2 women in their 70s) and 247 were injured (7 severely; 19 moderately). The affected persons were believed to have been trampled by people

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in the crowd, resulting in pressure on the chest and death from traumatic asphyxia^{14,15}.

Despite the large number of deaths from stampede accidents worldwide, the relationships between physical variables (e.g., compression site, compression load magnitude, and compression load time) associated with traumatic asphyxia and medical outcomes (such as respiratory failure, hypoxic encephalopathy, and death) are not well understood¹²⁻¹⁶. Clarifying these relationships could help in providing a medical basis for preventing deaths in crowd accidents, developing strategies for rescue, and establishing lifesaving measures.

This study estimated load conditions (load magnitude and loading time of the chest and abdomen) under which respiratory failure occurs in healthy women. In an actual accident, death is caused by respiratory failure. Needless to say, it is not possible to apply a load that would cause respiratory failure in studies of humans.

Previous studies applied compressive loads to the chest area alone in dogs or mice^{17,18}. The animals died when loads several times their body weight were applied, but it was difficult to identify the mechanisms that led to traumatic asphyxia. These previous studies of dogs and mice considered only the magnitudes of loads applied to the chest, loading time, and survival status. We hypothesized that in humans we would be able to simulate dyspnea preceding respiratory failure and death under conditions with much smaller load magnitudes than those applied in the animal studies. If persistent dyspnea were a potential cause or respiratory failure or death, the load conditions could predict traumatic asphyxia. We also hypothesized that loads applied to the abdomen would be more likely to cause respiratory failure than those applied to the chest, as compressive loads applied to the abdomen restrict diaphragm movement.

Methods

Participants

Because crowds typically comprise individuals of both sexes and varying ages, it is difficult to define a study sample. Moreover, ethical requirements limit use of children and elderly adults as participants, even though these groups accounted for most victims in past crowd accidents, because of their smaller physique and limited respiratory resistance^{1,6,7,14}. Therefore, this study enrolled young women as participants, as they generally have smaller physiques and less physical strength than men.

Fourteen healthy women participated in this study. Data obtained from an initial experiment conducted with

3 participants indicated that 9 participants would be required in order to detect differences between load patterns. The actual sample size was thus set at greater than 9. The variables assessed were tidal volume (VT) while standing, vital capacity (VC) while standing, VT while supine, and VC while supine.

Measurements

Participants were weighed in supine position with a dead weight load (10-kg iron plate; Irotec, Osaka, Japan) applied to the chest and abdomen (**Fig. 1**). Using 2 nylon belts (170 × 240 mm) placed over the front of the chest and abdomen of each participant, the weighted load (by gravity) was applied via chains connected to both ends of the belts.

The center of one belt was aligned with the center of the sternum, and the center of the other belt was aligned with the umbilicus. The load applied to the chest and abdomen was 0, 10, or 20 kg. A total of 8 load patterns (A-H) with various combinations of the weights were used, and each loading pattern was applied to the participants (**Table 1**).

Heart rate (beats/min) and SpO₂ were measured continuously, and brachial blood pressure (mm Hg) was measured intermittently every 2.5 min with a vital signs monitor (Propaq® Encore monitor; ZOLL Medical Corporation, Chelmsford, MA). Using a laser displacement measuring device (Intelligent-L Laser Sensor; Keyence Corporation, Osaka, Japan), we measured displacement of the anterior surface of the chest and abdomen in the vertical direction (anteroposterior displacement of the participant's body) associated with the applied load, respiratory excursion every 0.2 s, respiratory rate (breaths/min), and inspiratory and expiratory times.

In addition, VC (L) (intermittent measurement every 5 min [average of 3 measurements]) and VT (L) (intermittent measurement every 2.5 min [average of 5 measurements]) were measured with a spirometer (TKK 11510; Takei Scientific Instruments Co., Ltd., Niigata, Japan). Changes in these parameters over time were analyzed with the Friedman test. Subjective respiratory distress symptoms were evaluated by using the modified Borg Scale, which ranges from 0 (none) to 10 (extremely severe)¹⁹ (**Table 2**).

Loading (A-H) was performed after baseline measurement of each parameter for 2.5 min under conditions of no load. Loading time was set to a maximum of 20 min for each pattern, and measurements were continued for up to 5 min after unloading. When multiple load patterns were used for the same participant on the same

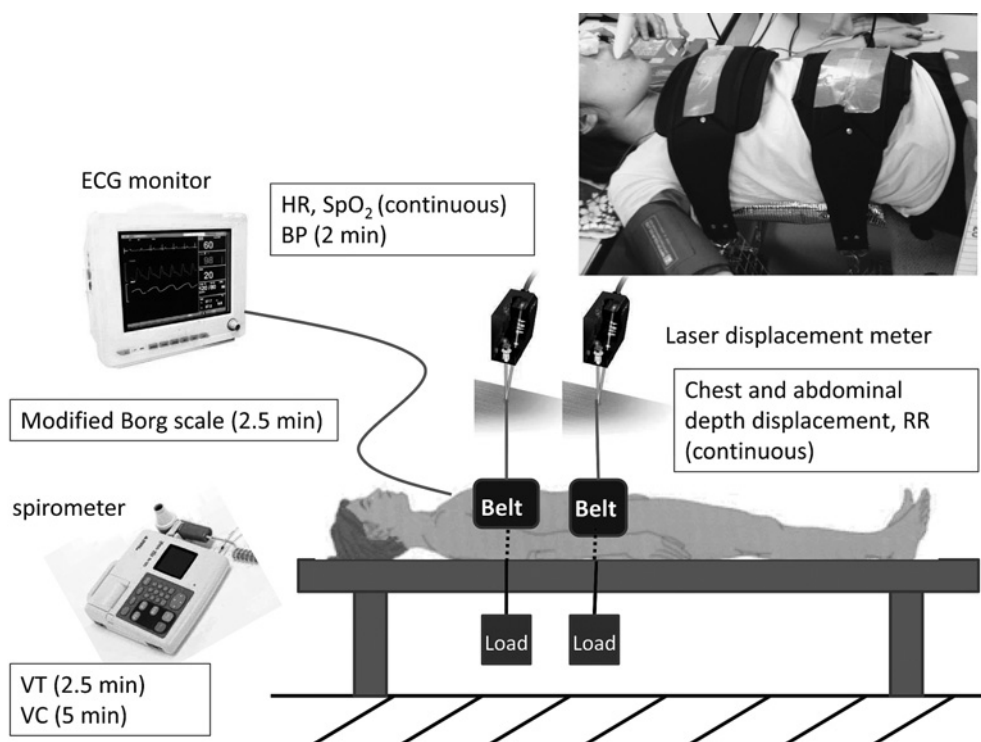


Fig. 1 Experimental setup for applying loads to the chest and abdomen. Loads (0, 10, and 20 kg) were applied by using two nylon belts (170 × 240 mm) to the front of the chest and abdomen of supine participants. An ECG monitor, SpO₂ oximeter, spirometer, and laser displacement meter were used to measure each variable. HR, heart rate; BP, blood pressure; SpO₂, percutaneous oxygen saturation; RR, respiratory rate; VT, tidal volume; VC, vital capacity.

Table 1 Chest and abdominal load patterns

Load pattern	Chest (kg)		
	0	10	20
Abdomen (kg)	0		
	No load (Control) [0%]	C (10, 0) [21%]	F (20, 0) [42%]
	10	D (10,10) [42%]	G (20,10) [63%]
	20	E (10,20) [63%]	H (20,20) [83%]

Loads of 0, 10, and 20 kg were applied to the chest and abdomen. Load patterns (A-H) (chest load (kg), abdominal load (kg)) are shown.

[]: Percentage of total loading to average weight (48 kg)

day, tests were performed with a minimum interval of 2 h to allow for recovery from respiratory muscle fatigue. The maximum number of patterns was limited to 3 per day.

Analysis

To identify load patterns and loading times that could lead to respiratory failure, we calculated T_i/T_{tot} ratio, which is the ratio of inspiratory time (T_i) to total time for 1 respiratory cycle (T_{tot}); VT/VC , which is the ratio of tidal volume (VT) to vital capacity (VC); and the Breathing Intolerance Index (BITI)²⁰, which is an index of respiratory muscle fatigue expressed as the product of T_i/T_{tot}

and VT/VC . Previous studies reported that a normal BITI value is <0.05 , and a value of ≥ 0.15 is within the critical range, at which respiratory failure occurs within about 45 min^{20,21}.

Changes in HR, sBP, dBP, SpO₂, RR, tidal volume, minute volume, VT, T_i/T_{tot} , VT/VC , and BITI from baseline (no load) until 20 min of loading were analyzed for each load pattern with the Friedman test.

To clarify the relationship between conditions that can lead to respiratory failure and VC and subjective degree of respiratory distress, correlations between BITI and VC and between BITI and modified Borg Scale score were

Table 2 Modified Borg dyspnea scale

Point (s)	Degree of breathlessness
0	None
0.5	Very, very slight (just noticeable)
1	Very slight
2	Slight
3	Moderate
4	Somewhat severe
5	Severe
6	
7	Very severe
8	
9	Extremely severe (almost max)
10	Maximal

Subjective respiratory distress symptoms over time were recorded with the modified Borg dyspnea scale.

analyzed. Furthermore, to identify chest or abdominal loads that exerted a greater effect on respiratory failure, change in BITI when the abdominal load was kept constant and chest load was changed, and when the chest load was kept constant and the abdominal load changed, were analyzed with 1-way analysis of variance and the Kruskal-Wallis test.

Data are expressed as the median of all measurements for each load pattern. Statistical analysis was performed with SPSS Statistics version 16.0 (SPSS, Inc., Chicago, IL, USA). Significance was set at $p < 0.05$.

Ethical Issues

Participants gave written informed consent to participate after being given a full explanation of the objectives, significance, and safety measures of the study. This study was approved by the ethics committee of Nippon Medical School, Chiba Hokusoh Hospital, Chiba, Japan (approval number 572). During the approval process, input was sought from clinical physicians, forensic pathologists, nurses, the general public, and others. Approval was given by the committee after careful, multidisciplinary investigation of loads applied to the chest and abdomen, including load magnitude and loading time, and of standards for study termination. The study was conducted in accordance with the Declaration of Helsinki. Participants were university students recruited by public invitation, and none were directly acquainted with the researchers. Participants were paid for the time they spent participating in the study (including travel time). To ensure the safety of participants, a physician with no prior interest in this research performed a physical examination, height and weight measurement, and chest X-ray examination and obtained a full medical history.

The physician, together with other staff, was always present near the participant during load application and monitored vital signs and subjective and objective findings. Preparations were made to remove the load immediately and administer appropriate medical treatment. The experiment was stopped if any safety problem arose, if the participant requested to stop, if the modified Borg Scale was 8 or higher, or if the accompanying physician deemed it necessary to stop.

To enable rapid access to emergency medical procedures were the need to arise, the experiment was conducted next to the emergency department of Nippon Medical School, Chiba Hokusoh Hospital.

The participants were observed by physicians for 2 h after completing the experiments and were further examined at 1 week and 1 month after the experiments, to identify any changes in their physical status or other relevant characteristics. No abnormalities were observed in any participant.

Results

In total, 14 healthy women participated in this study (age [SD] 27.5 [21.5-28.0] years, height 159.5 [153.5-164.5] cm, body weight 48.0 [43.0-53.5] kg, body mass index [BMI] 19.1 [17.3-20.6]). Tidal volume (VT) while standing was 0.73 (0.62-0.93) L, vital capacity (VC) while standing was 2.99 (2.79-3.46) L, VT while supine was 0.54 (0.41-0.86) L, and VC while supine was 2.75 (2.34-3.04) L (Table 3).

Heart rate (Fig. 2), systolic blood pressure (Fig. 3a), SpO₂ (Fig. 4), and respiratory minute volume did not change over time in any load pattern. Diastolic blood pressure significantly increased over time in pattern H (Fig. 3b). Respiratory rate significantly increased over time in patterns D, E, F, G, and H (Fig. 5). VT significantly decreased over time in patterns B, E, G, and H (Fig. 6). VC significantly decreased over time in patterns C, D, E, F, G, and H (Fig. 7).

BITI increased significantly over time in patterns G and H. Linear regression lines are expressed by the equations $y = 0.0025x + 0.1146$ (pattern G) ($p = 0.022$, $R^2 = 0.44$) and $y = 0.0027x + 0.1440$ (pattern H) ($p = 0.021$, $R^2 = 0.43$); BITI was ≥ 0.15 at about 14 min after the start of loading in pattern G and about 2 min after the start of loading in pattern H (Fig. 8).

BITI and VC were significantly inversely correlated ($p = 0.000$, Pearson correlation coefficient = 0.509), as represented by the regression equation $y(\text{BITI}) = -0.1456x(\text{VC}) + 0.4188$ ($R = 0.51$, $R^2 = 0.26$). When BITI was ≥ 0.15 , the critical threshold value, VC, was < 1.85 L (Fig. 9).

Table 3 Basic characteristics of participants (median [IQR])

		Median [IQR]
Age (years)		27.5 [21.5-28.0]
Height (cm)		159.5 [153.5-164.5]
Weight (kg)		48.0 [43.0-53.5]
Body mass index		19.1 [17.3-20.6]
Standing	Tidal volume (L)	0.73 [0.62-0.93]
	Vital Capacity (L)	2.99 [2.79-3.46]
Supine	Tidal volume (L)	0.54 [0.41-0.86]
	Vital Capacity (L)	2.75 [2.34-3.04]

The study participants were 14 healthy women in their 20s. Age, height (cm), weight (kg), BMI, tidal volume (L) and vital capacity (L) while standing and supine are shown.

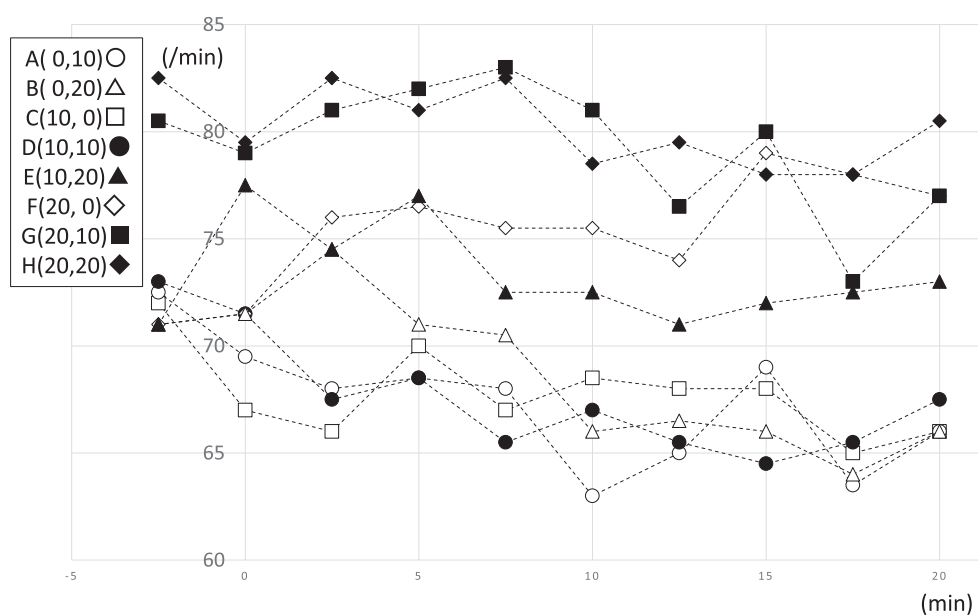


Fig. 2 Change over time in heart rate (/min). No significant changes over time were observed for any pattern. Changes over time from baseline (no load) until 20 min of loading were analyzed for each load pattern with the Friedman test.

BITI and modified Borg scale score were weakly correlated ($p = 0.000$, Pearson correlation coefficient = 0.294) with a regression equation of $y(\text{BITI}) = 0.006x$ (modified Borg scale) + 0.100 ($R = 0.29$, $R^2 = 0.09$). When BITI was ≥ 0.15 , the modified Borg scale score was ≥ 8.3 (indicating very severe to extremely severe [almost maximal] dyspnea) (Fig. 10).

No significant difference was observed in BITI when abdominal load was increased while chest load remained constant (0, 10, and 20 kg). In contrast, BITI significantly increased ($p = 0.048$, 0.000, and 0.008, respectively) when chest load was increased while abdominal load remained constant (0, 10, and 20 kg) (Fig. 11).

Discussion

Traumatic asphyxia is a major cause of death in human stampede accidents. The 2010 Love Parade accident in Germany resulted in the deaths of 21 people in their 20s to 40s (8 men, 13 women)⁶. In 2010, 347 people were killed at the Khmer Water Festival in Cambodia⁶. Crowd accidents during the Hajj, the annual Islamic pilgrimage to Mecca, result in deaths every few years: 1,426 in 1990, 270 in 1994, 119 in 1998, 35 in 2001, 14 in 2003, 251 in 2004, and 380 in 2006⁷. Traumatic asphyxia was likely the major cause of death in all these accidents, but no careful examinations, including autopsies, were conducted to identify causes of death^{1,6,8,13}.

Here, we applied loads to the human chest and abdomen to identify the mechanism involved in deaths dur-

Mechanism of traumatic asphyxia

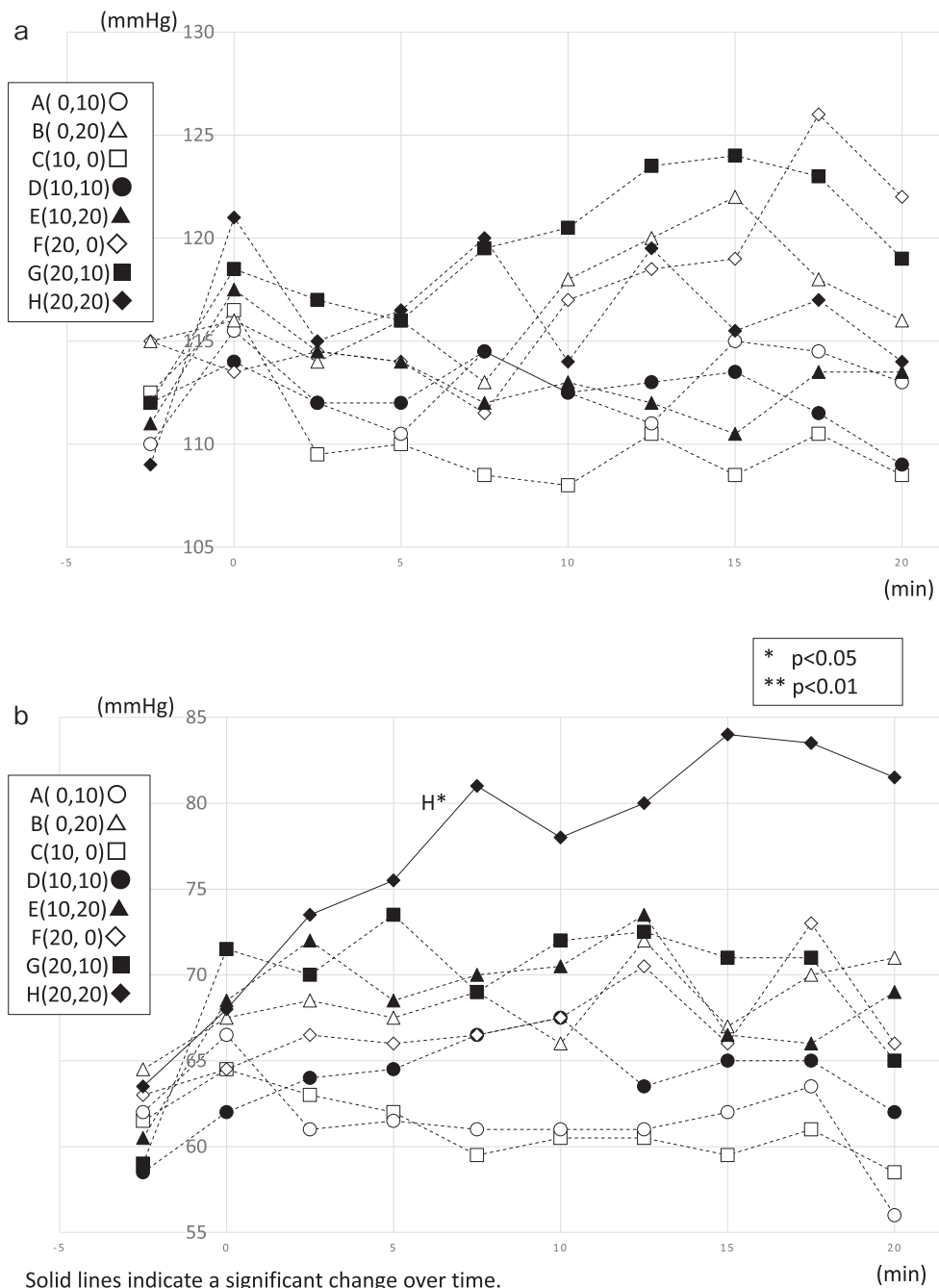


Fig. 3 a. Change over time in systolic blood pressure (mm Hg). No significant changes over time were observed for any pattern. Changes over time from baseline (no load) until 20 min of loading were analyzed for each load pattern with the Friedman test.
 b. Change over time in diastolic blood pressure (mm Hg). Diastolic blood pressure increased over time in pattern H. Changes over time from baseline (no load) until 20 min of loading were analyzed for each load pattern with the Friedman test.

ing stampede accidents. Before conducting this human study, we attempted to conduct the experiments with pigs. Anesthesia was required to secure the pigs on a bench in the supine position. However, even a low dose of anesthesia affected respiration, causing respiratory depression. It was extremely difficult to control the depth of anesthesia so that the pigs would continue normal,

calm spontaneous breathing on a bench. Thus, we halted the pig experiments after studying 8 pigs.

The present findings suggest that respiratory muscle fatigue increased over time when the chest load exceeded 20 kg and abdominal load exceeded 10 kg and that this could lead to respiratory failure. When a load equivalent to approximately 60% of the participants' body weight

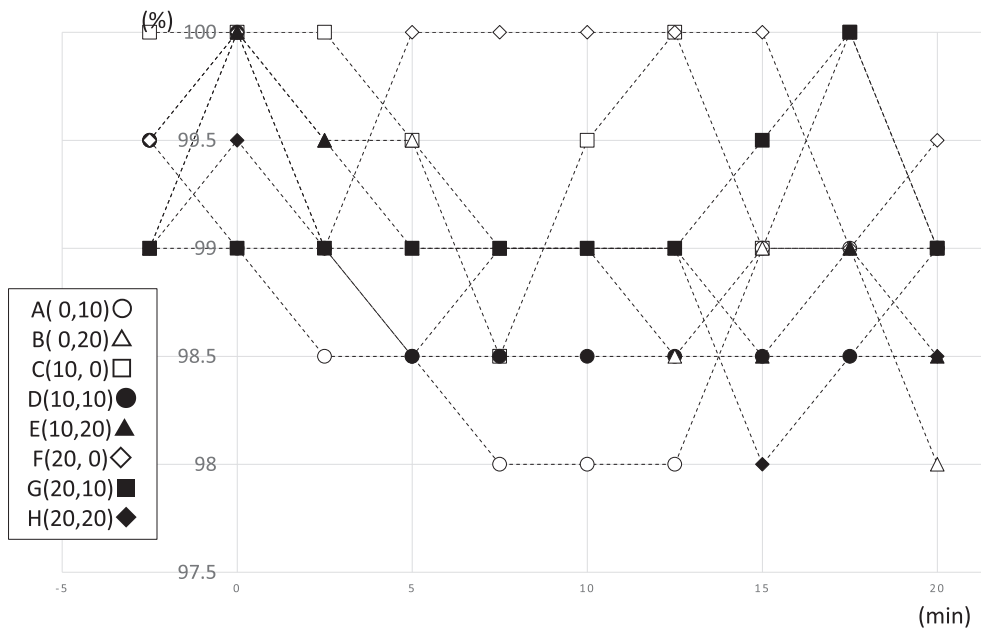


Fig. 4 Change over time in SpO₂ (%). No significant changes over time were observed for any pattern. Changes over time from baseline (no load) until 20 min of loading were analyzed for each load pattern with the Friedman test.

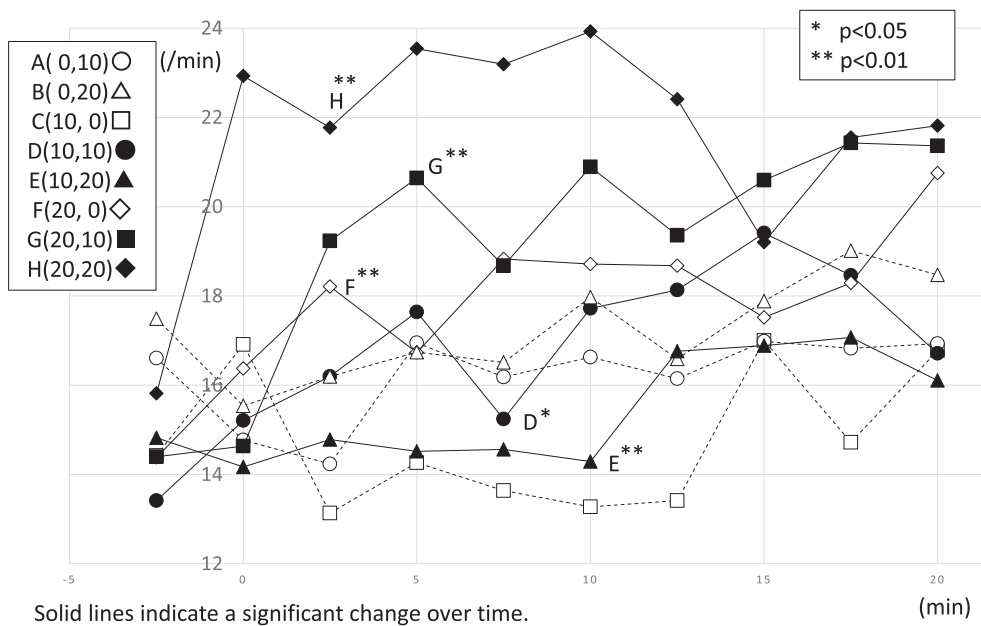


Fig. 5 Change over time in respiratory rate (breaths/min). Respiratory rate increased in patterns D, E, F, and H. Changes over time from baseline (no load) until 20 min of loading were analyzed for each load pattern with the Friedman test. (*p < 0.05, **p < 0.01)

was applied to the thoracoabdominal region, analysis of BITI clearly showed that respiratory failure was inevitable within 1 h from the start of loading. In addition, with thoracoabdominal compression, a VC of ≤ 1.85 L and a modified Borg scale score ≥ 8.3 was equivalent to a BITI of ≥ 0.15 , which is considered a state of dangerous respiratory muscle fatigue. Furthermore, respiratory muscle

fatigue rose with increasing chest load under a constant abdominal load. However, respiratory muscle fatigue did not increase as abdominal load was increased under a constant chest load.

A previous chest compression study reported that all dogs survived for longer than 60 min under a load of twice body weight, but 75% died within 10 min under a

Mechanism of traumatic asphyxia

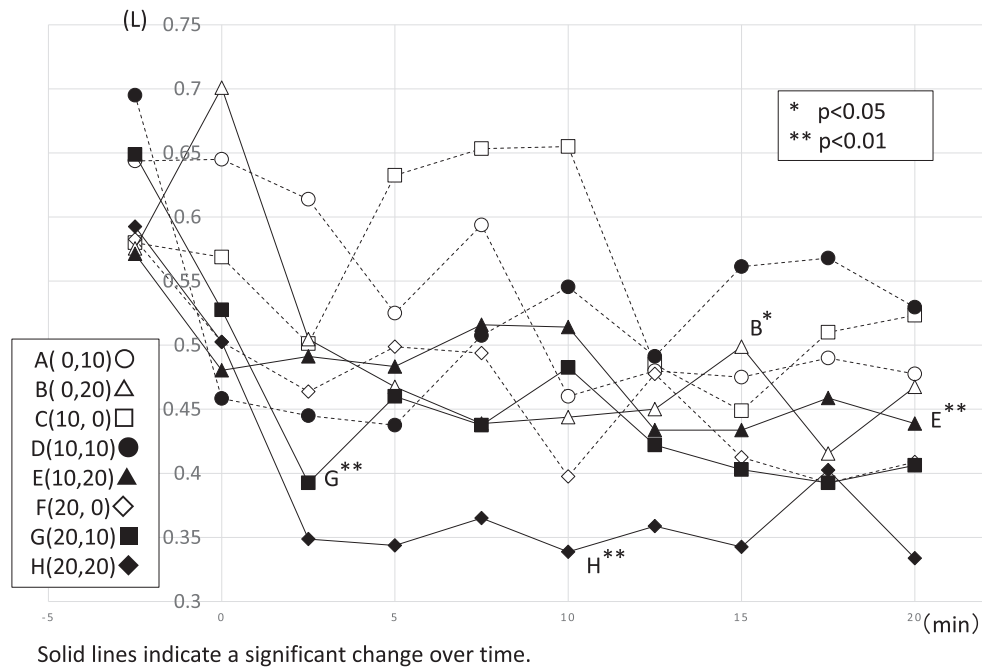


Fig. 6 Change over time in tidal volume (L). Tidal volume decreased in patterns B, E, G, and H. Changes over time from baseline (no load) until 20 min of loading were analyzed for each load pattern with the Friedman test. (*p < 0.05, **p < 0.01)

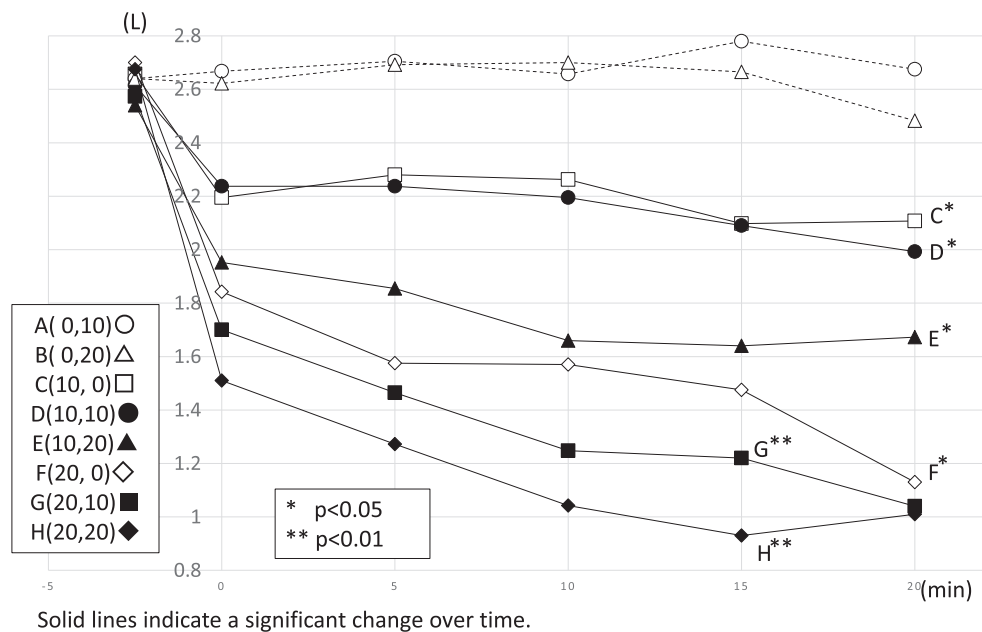


Fig. 7 Change over time in vital capacity (L). Vital capacity decreased in patterns C, D, E, F, G, and H. Changes over time from baseline (no load) until 20 min of loading were analyzed for each load pattern with the Friedman test. (*p < 0.05, **p < 0.01)

load of 4 times body weight. All dogs died within 10 min under a load of 5 times body weight¹⁹. Another study reported that, after chest compression of mice (body weight: 480-550 g), all mice survived more than 60 min under a load of twice body weight, but all mice died within 40 min under a load of 3 times body weight and

within 10 min under a load of more than 4 times body weight²⁰. Taken together, these findings show that compression load magnitude and duration are important variables in the mechanism of traumatic asphyxia. However, it is unclear how these variables affect traumatic asphyxia in humans.

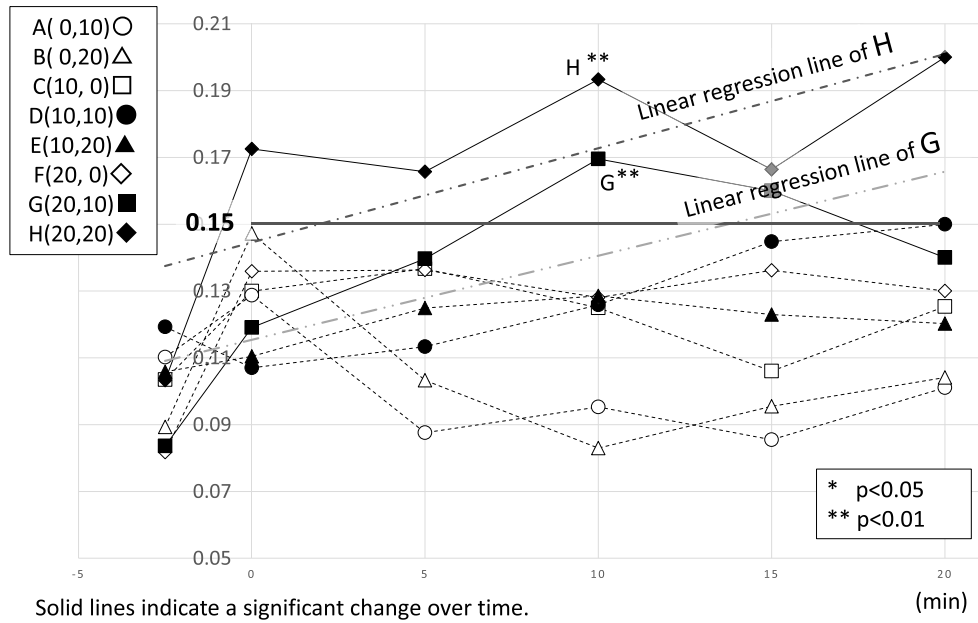


Fig. 8 Change over time in BITI. BITI increased in patterns G and H and reached the critical range (≥ 0.15) at 14 min and 2 min after the start of loading, respectively. BITI: Breathing Intolerance Index. Changes over time from baseline (no load) until 20 min of loading were analyzed for each load pattern with the Friedman test. (* $p < 0.05$, ** $p < 0.01$)

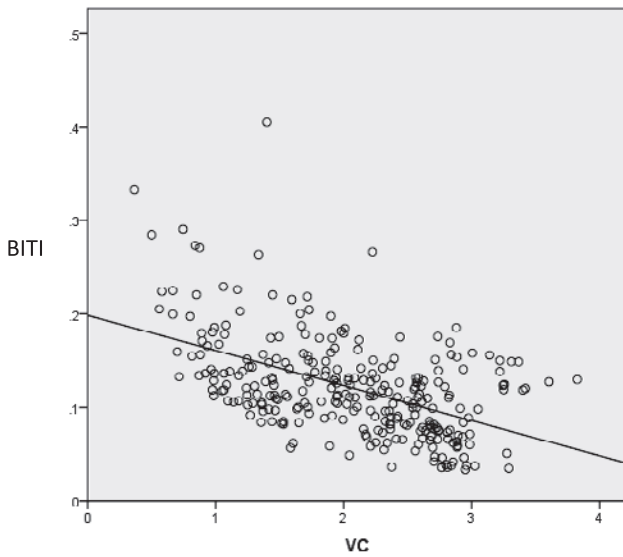


Fig. 9 Inverse correlation between BITI and vital capacity (VC). BITI and vital capacity (VC) were inversely correlated ($p = 0.000$); the regression equation was $y(\text{BITI}) = -0.1456x(\text{VC}) + 0.4188$ ($R = 0.51$, $R^2 = 0.26$). BITI: Breathing Intolerance Index.

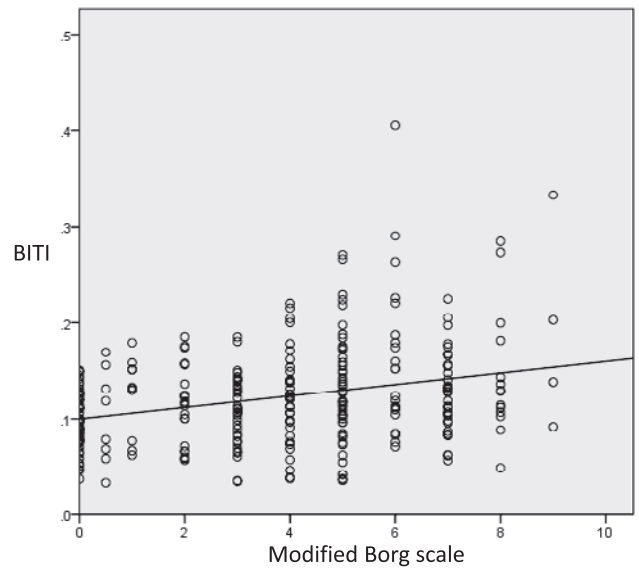


Fig. 10 Correlation between BITI and modified Borg scale score. BITI and Borg scale were positively correlated ($p = 0.000$); the regression equation was $y(\text{BITI}) = 0.006x(\text{Borg scale score}) + 0.100$ ($R = 0.29$, $R^2 = 0.09$). BITI: Breathing Intolerance Index.

Although load was determined as a percentage of body weight in these animal studies^{19,20}, absolute load calculations are considered more useful for implementing measures to prevent actual crowd accidents. Thus, absolute loads (0, 10, and 20 kg) were used in this study.

Ideally, an even more detailed analysis would be possi-

ble if loads were set at 5-kg intervals and more load patterns were studied. However, overly prolonging the study duration would be burdensome to participants and would affect study participation and completion; consequently, only 8 load patterns were analyzed in this study.

Bellemare and Grassino studied respiratory tolerance

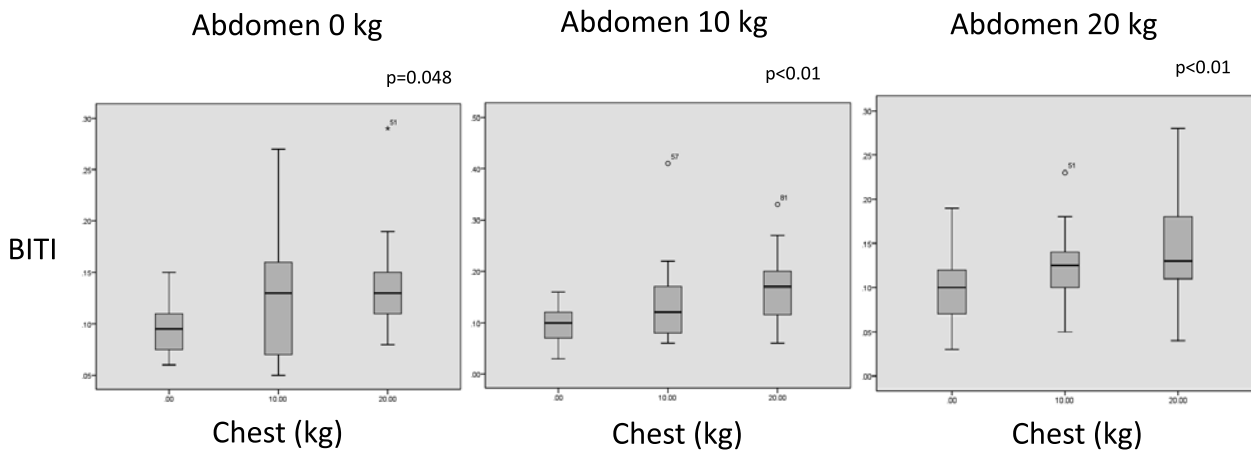


Fig. 11 Changes in BITI with change in chest load and constant abdominal load; abdominal load was 0, 10, or 20 kg. BITI increased significantly when the chest load was increased ($p = 0.048, 0.000, \text{ and } 0.008$, respectively) (Kruskal-Wallis test). BITI: Breathing Intolerance Index.

(respiratory muscle fatigue) of the diaphragm and found that respiratory failure could be predicted by the diaphragm time tension index (TT_{di}), which is calculated by using inspiratory time (T_i), total respiratory cycle time (T_{tot}), transdiaphragmatic pressure (P_{di}) at rest ventilation, and maximum transdiaphragmatic pressure (P_{dimax}). Respiratory failure occurred about 45 min after reaching a TT_{di} of ≥ 0.15 ²¹⁻²³.

$$TT_{di} = (T_i/T_{tot}) \cdot (P_{di}/P_{dimax})^{21} \quad (1)$$

However, measurement of P_{di} and P_{dimax} requires insertion of an invasive gastroesophageal balloon catheter. These variables are therefore difficult to measure in human experiments. Koga et al. found that BITI could be calculated by using T_i , T_{tot} , VT, and VC.

$$BITI = (T_i/T_{tot}) \cdot (VT/VC)^{20,24} \quad (2)$$

Equation (2) substitutes the ratio VT/VC , which can be measured noninvasively for P_{di}/P_{dimax} in equation (1)^{20,25}. BITI is expressed as the reciprocal of respiratory endurance and is believed to reflect both the respiratory reserve of the diaphragm alone and fatigue of all muscles related to breathing²⁰.

The normal BITI value is assumed to be < 0.05 , similar to TT_{di} . A BITI of ≥ 0.15 is believed to be in the critical range, after which respiratory failure occurs in approximately 45 min²⁰. In recent years, BITI has been used as an indication for withdrawal of mechanical ventilation in pediatric intensive care units^{25,26}.

This study showed that BITI reaches the critical range at 14 min after loading in pattern G and at 2 min after loading in pattern H. This suggests that these load patterns would lead to respiratory failure within 59 min and 47 min after loading, respectively.

Previous studies of traumatic asphyxia used only chest

compression^{17,18,27,28}, and no study considered compression of the abdomen or fatigue of the diaphragm. However, in actual crowd accidents, both the abdomen and chest are under load. This might limit the movement of the diaphragm, the most important respiratory muscle, and thus adversely affect respiratory dynamics²⁹. This study is the first to examine the effects of combined chest and abdominal loading on respiratory failure.

Our findings suggest that healthy women in their 20s with a load pattern of 20 kg on the chest and 10 kg on the abdomen (pattern G) or 20 kg on the chest and 20 kg on the abdomen (pattern H) could go into respiratory failure within 1 h, even when the total load magnitude is less than their body weight (approximately 60%-80% of body weight). These loads are smaller than those in previous reports^{17,18,27,28}. This finding may be attributable to the presence of loads on both the abdomen and chest, which is similar to the conditions of actual crowd accidents. Moreover, a $VC \leq 1.85$ L and modified Borg scale score of ≥ 8.3 indicated a very severe to extremely severe (almost maximal) state equivalent to reaching the critical range. Under such conditions, BITI is ≥ 0.15 based on the correlations between BITI and VC and between BITI and the modified Borg scale score.

For the load range used in this study (maximum load of 20 kg on both the chest and abdomen), an increase in chest load was significantly associated with an increase in BITI. However, BITI did not change as abdominal load was increased, which suggests that, in crowd accidents, the effect on respiratory dynamics starts to appear from compression above a certain load magnitude on the chest (20 kg in this study) and that this is modified by abdominal load.

This study has some limitations. First, because the participants were humans, enrolling a large sample was challenging. Second, data on children and elderly adults, who are the most vulnerable in actual disaster situations, were unavailable because the participants were limited to healthy young women, to ensure the safety of the experiments. Third, only female participants were used because they have a smaller physique and lower respiratory reserve capacity than men; thus, the effects of sex differences were not assessed. Nevertheless, this study provides data applicable to the safety of people who would be vulnerable in a crowd accident. Fourth, loading experiments were performed only in the supine position because the loading environment was designed to use the force of gravity alone; measurements in other positions, such as standing or prone positions, could not be performed. In an actual disaster, however, it is necessary to consider the effects of posture on breathing^{30,31}. Fifth, although the modified Borg scale was used to grade breathing difficulty, this represents only the degree of subjective respiratory distress and is not objective. Finally, although collection of arterial blood gas data is particularly useful for objectively defining respiratory failure, invasive arterial blood sampling was not performed in this study.

To our knowledge, this is the first study to clarify the relationship between load magnitude, loading time, and respiratory failure in humans. The relationship between VC and subjective breathing difficulty for predicting respiratory failure was also clarified.

Most victims of traumatic asphyxia in crowd accidents are children or elderly adults. Respiration resistance is lower in children with high thoracic extensibility and older people with low respiratory reserve capacity than in the present adult female participants. Thus, respiratory failure and death may occur earlier when children or older adults are subjected to an equivalent load in relation to body weight.

Clarifying the mechanisms of traumatic asphyxia, including determining the load magnitude and duration of thoracoabdominal compression leading to traumatic asphyxia, could help prevent traumatic asphyxia in crowd accidents through space design based on crowd control simulation³²⁻³⁴ and restrictions on entering the venue. It could also facilitate rescue activities and emergency medical services at accident sites.

Because victims of crowd disasters where rescue efforts are ongoing may experience respiratory failure due to respiratory muscle fatigue over time, the present results

may aid in developing new rescue strategies, such as emergency response plans and approaches for decision-making regarding medical interventions during rescue efforts.

Conclusion

A model experiment of traumatic asphyxia caused by thoracoabdominal compression was performed in healthy women. The findings suggest that respiratory failure would occur within 1 h because of fatigue of the respiratory muscles, even when the load on the chest and abdomen was approximately 60% of body weight. The pathophysiology of respiratory failure was more strongly affected by increasing chest load than by increasing abdominal load. Under a chest and abdominal loading environment, a VC of ≤ 1.85 L and a modified Borg scale score of ≥ 8.3 can be used as indices to predict respiratory failure.

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Conflict of Interest: The authors declare that they have no conflicts of interest.

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